The Long-Term Economic Costs of the Great London Smog

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This paper studies the long-term economic effects of early exposure to the Great London Smog of 1952. Cohorts born in London are tracked for up to sixty years using the Office of National Statistics Longitudinal Study. Exposure to the four day smog reduced the size of the surviving cohort by 2% and caused lasting damage to human capital accumulation, employment, hours of work, and propensity to develop cancer. (*JEL* : Q53, I12, I18)

1 Introduction

This paper studies the long-term labour market effects of exposure to the Great London Smog for cohorts that were *in utero* or young infants in London at the time.

The Great Smog began on the fifth of December, 1952, when a high pressure weather system settled over London, creating an envelope of cold air that prevented the normal dispersion of atmospheric pollution. A thick ground-level smog formed over the city that lasted for a further four days. The Ministry of Health (1954) attributed 4,000 premature deaths to exposure to the smog, a figure that has been revised to 12,000 in recent studies (Bell & Davis, 2001). Parliament responded with Clean Air Act of 1956, drafted with the goal of preventing any further smogs in London.

A growing body of evidence has linked various early-life exposures to poor health and labour market performance in later life (Almond & Currie, 2011.) Additionally,

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three papers have linked exposures to urban atmospheric pollution and long-term labour market outcomes (Sanders, 2012; Isen *et al*, 2017; Bharadwaj *et al*, 2016). The Great London Smog was quite different from the context of these studies. First, because the Great London Smog was only a few days long, while existing evidence relates to long-term average exposures. Second, because pollution exposures in December '52 were particularly severe - more comparable to severe smog events in Mumbai or Beijing than to exposures in modern day London. To my knowledge, this is the first study of the long-term economic effects of early or *in utero* exposure to the London Smog, or of any similar urban smog[1].

Many of the hypothesised labour market effects of early exposure to pollution can take decades to manifest. An advantage of studying the Great London Smog is that those affected can be observed for almost their entire working lives. In the analysis, those who were *in utero* or infants in London during the Smog are traced by their year and borough[2] of birth, and observed using the Office of National Statistics Longitudinal Study every decade from 1971, when these cohorts are around twenty, to 2011, when these cohorts are around sixty. Administrative data linked to the study makes it possible to observe effects on human capital accumulation, employment over the working life, working hours, absence from work due to a permanent illness, cancer registrations, and mortality.

Those *in utero* during the Smog would have been born in 1953. An analysis using historical data from the Registrar General showed around 1280 fewer births in that year than would be expected, or a reduction of 2% in the size of the *in utero* cohort. Those observed later in life were 3% less likely to hold a degree-level qualification. There was no effect on unemployment, but there was a significant effect on hours worked in the early career, with those *in utero* during the Smog working 8 fewer hours a week and those who were infants during the Smog working 4 fewer hours a week. There was no effect on cancer registration for those who were *in utero* during the Smog but those who were infants (and so exposed to the Smog directly) were 3% more likely to be diagnosed with cancer. Lastly, the treated cohorts were both 2% less likely to die in sample - suggesting that those not surviving to be observed would have died before the age of sixty, in the absence of the Smog.

The effect of the Great London Smog on adult mortality is what caused the U.K. Parliament to take the costs of atmospheric pollution seriously. The results of this paper suggest that the long-term health of those who were *in utero* or infants during the smog was also affected, with deleterious effects on their working lives. Had these future costs been known, exposure to the Great London smog could *in principle* have been avoided

[1] Bharadwaj, Graff Ziven, Mullins, and Neidell (2016) study the effect of early exposure to the Great London Smog on the development of asthma using data from the English Longitudinal Study of Aging. The data do not allow a large sample size - 42 people in the data were exposed in utero, and 15 at less than one year old - but the estimated coefficients map well with the pattern of Smogs in London, with higher reports of Asthma among London cohorts affected by the 1948 and 1952 Smogs.

[2] Greater London is divided into 32 Boroughs, which average around 40 square kilometres in size, and had average populations of around 100,000 at the time of the smog. In the E.U. categorisation, one or two Boroughs form a NUTS3 region.

by vulnerable populations. The weather event that caused it had been forecasted and daily pollution meters recorded the severity of the smog. The duration of such events was known, and avoiding London for less than a week might have been economically feasible. This is in contrast to normal, ambient levels of pollution, which were high in London and difficult to avoid without a permanent move elsewhere.

If some of the costs of high ambient levels of pollution come from short and forecastable events, such London's smogs, avoidance would be a sensible strategy. The Great London Smog was a unique event, but in some important dimensions, such as its duration and levels of particulate pollution, it bears comparison with smogs in present day industrialising economies. To give one extreme example, the 2013 smog in North-East China lasted five days and saw hourly pollution levels of over 1,000 $\mu g/m^3$ PM2.5 in Harbin, a city of more than five million people. It is for future research to determine the long-term costs of these modern events, but pollution forecasting is now cheap and effective, and the potential benefits of avoidance - if the Great London Smog is any guide - could be high.

2 Background

This section provides background on how early exposures to pollution *in utero* or as an infant might affect long-term labour market outcomes, and a review of the empirical literature on this channel.

2.1 Mechanism: How pollution can affect long-term outcomes

Pollution affects labour market outcomes primarily through the health channel. A key source of damage from arises from oxidative stress[3]: Free radicals from pollutants attack cells at the molecular level, destroying proteins, lipids (fats), and DNA. This process can kill or damage cells and is, in fact, an important part of how the immune system destroys pathogens. The main components of urban pollution either *are* free radicals (CO, NO_x) or produce them in the body (O_3, PM).

The body has both active and passive defences that respond to exposure. Some parts of the body - such as the lung lining fluid - naturally contain anti-oxidants that can protect critical systems from a degree of exposure by neutralising free radicals before they cause damage. In contrast, the effects of exposure can be magnified by the body's immune response. Inflammatory cells flood affected and unaffected areas with free radicals which, in the absence of pathogens, attack native cells. This is one mechanism by which pollution exposure could result in damage to the heart, and other critical systems.

Health effects can emerge quickly. Ozone is very reactive, and exposure can result in adverse health effects in less than an hour, while effects from particulate matter have been recorded after less then four hours exposure (Rundel, 2012). The second round of

[3]Unless otherwise stated, information on oxidative stress is from Kelly (2003), who surveys the medical and epidemiology literatures on the health effects of pollution through the oxidative stress channel.

oxidative stress from the body's immune response can last from six to eighteen hours after exposure.

Kannan et al (2006) discuss the channels through which fetal exposure to pollution can affect health. One of the most well documented is the effect of polycyclic aromatic hydrocarbons (PAHs), which are found on particulate matter. PAHs are able to cross the placenta and form free radicals when metabolised, exposing the foetus to oxidative stress. Exposure in the first trimester, when the neural tube is formed, is expected to be most harmful. Exposure to PAHs has been linked to fetal mortality and – among those surviving to term – low birthweight, and smaller head circumference (Jayachandran, 2009).

2.2 Early exposure to pollution: short-term outcomes

A major concern in studying the effects of pollution on health is accounting for non-random assignment (Graff Zivin & Neidel 2013). The dominant approach in the economics literature is to study plausibly exogenous variation in pollution exposure from external shocks such as recessions or policy changes (Chay and Greenstone, 2003), natural disasters (Jayachandran 2009), shocks to emissions (Pope 1986, Schlenker & Walker 2016) or external factors affecting location (Lleras-Muney 2010)[4].

Studying the effects of atmospheric pollution on fetal loss presents the additional challenge that fetal losses are often under-reported and, should they occur early in the pregnancy, can even go unnoticed by the mother. Sanders and Stoecker (2011) take an indirect approach, exploiting the Trivers-Willard hypothesis that male foetuses are more vulnerable to external shocks than females. Using plausibly exogenous variation from the 1970 Clean Air Act and data from a 50% sample of U.S. birth certificates, the authors find that a one standard deviation drop in annual average particulates reduces the percentage of male live births by 3.1%. Jayachandran (2009) provides information on the effects of exposure to high levels of particulates. She overcomes the under-reporting of stillbirths by focusing instead on recorded births. Exploiting spatial and temporal variation from a wildfire that swept through Indonesia, the paper finds that exposure to a short, severe pollution shock led to 15,600 fewer births in subsequent months[5].

There is a growing literature on the effects of fetal exposure to pollution on health at birth. A fetal insult can have two effects on the relative health of a birth cohort. Exposure to pollution could lead to 'scarring' effects, whereby fetal exposure harms the health of each individual. Exposure could also lead to 'culling' effects, whereby the fetal insult affects the composition of the surviving cohort. When it is the strongest that survive a shock, these effects combine to make surviving cohorts appear healthier than they are[6]. Bozzoli, Deaton and Qintana-Domeque (2009) encounter this effect when

[4]This approach is also taken by some papers in the epidemiology literature - a notable example is Pope (1986), who studies the effect of the closure of a major emitter.

[5]Another paper taking this approach is Kwawand et al 2015, who study the effects of wildfires in California.

[6]See Almond and Currie (2011) for a fuller discussion of 'culling' in papers studying the effects of fetal shocks.

studying the cross-country link between child mortality and adult height. The authors find the expected relationship among most countries, but find that child mortality is associated with an *increase* in the height of the surviving population in the poorest countries, where mortality was particularly high.

Nonetheless, there is strong evidence that fetal exposure to pollution affects infant health and mortality[7]. In utero exposure to pollution has been linked to prematurity, low birth weight and size (Liuchinger et al 2014, Yang & Chou 2016, Cho et al 2013). Currie and Walker (2011) study the effects of air pollution caused by traffic using variation from the introduction of the EZ-Pass scheme in New Jersey and Pennsylvania. This scheme allowed drivers to pass through toll gates without stopping, and resulted in a sharp reduction in carbon monoxide pollution in residential areas close to the tolls. The paper uses a differences-in-differences strategy, comparing the change in infant health of those *in utero* close to highway tolls to those *in utero* close to other parts of the highway system. Their results show that the introduction of the EZ-Pass scheme resulted in around an 11% reduction in prematurity, and a 12% reduction in low birth weight - a common proxy of infant health.

Both *in utero* and neonatal exposure to pollution have been linked to increased infant mortality (Currie & Neidell 2005, Knittel et al 2011, Schlenker & Walker 2015, Clay et al 2016, Arceo et al 2016, Greenstone & Hanna 2014, Tanaka 2015, Khawand et al 2015) though it is often difficult to distinguish between the effects of in-utero and neonatal exposure, which tend to be highly correlated. Chay and Greenstone (2003) use variation in pollution caused by the 1981-82 U.S. recession to study the effects of particulate pollution on infant mortality. They find that a 1% decrease in pollution in a county results in a 0.35% reduction in the infant mortality rate. The strongest effects were found for infants less than one month old, suggesting that fetal exposure was an important factor. There is much less evidence from countries with high levels of ambient pollution, due mostly to the difficulty in obtaining information on health and pollution. Arceo-Gomez, Hanna & Oliva (2016) gather ten years of weekly data on health for forty eight municipalities in Mexico City, where data for pollution is also available. The authors adopt an IV strategy using temperature inversions - which prevent the dispersion of atmospheric pollution - as an instrument for exposure. The IV estimates show that a $1\mu g/m^3$ increase in particulates results in 0.24 infant deaths per 100,000 births - a health effect similar to those found in the literature on the United States. Greenstone and Hanna (2014) construct a database of infant health and pollution levels in India in order to study the effectiveness of environmental regulations. The authors also test the effects of the most successful of the reforms, which promoted the use of catalytic converters, on infant mortality. Their results were suggestive of a decline in infant mortality, but were not statistically significant.

[7]See Zivin & Neidell (2013) for a survey of the health effects of pollution, including the effects of fetal exposure on infant mortality. Currie and Vogl (2013) provide an overview of the long-term effects of early shocks in developing countries, including those from atmospheric pollution.

2.3 *Early exposure to pollution: long-term outcomes*

To the best of my knowledge, there is currently no evidence on the long-term economic effects of exposure to an urban smog, such as that in London in 1952[8]. There is also very little evidence on the longer-term effects of fetal exposure to long-term levels of pollution due to the difficulty in obtaining information on place of birth for individuals observed as adults[9]. I am aware of three studies of longer-term effects of exposure - though none track individuals for long enough to observe the more serious health effects (such as cardiovascular damage or cancer) that can result from pollution exposure.

The earliest, Sanders (2012) studies the effect of fetal exposure to pollution on performance in high school, using administrative data from Texas. Information on place of birth is not available, and *in utero* assignment of pollution is based on the county in which the high school is located. Exposure is calculated using county-level data on total suspended particulate matter, and is instrumented using county-level changes in relative manufacturing employment. The study finds that a standard deviation decrease in particulates is associated with a 2% increase in grades using OLS, and in a 6% increase using IV.

Isen, Rossin-Slater and Walker (2017) use linked administrative data from the U.S. census to investigate the effects of fetal exposure to particulates on income at the age of thirty. To identify the effect, the authors exploit a sharp drop in atmospheric pollution that followed the implementation of the 1970 Clean Air Act. Their results indicate that a 10 unit decrease in PM10 particulates resulted in a 1% increase in earnings for individuals aged 29-30, mostly driven by a drop in labour force participation.

Lastly, Bharadwaj, Gibson, Graff Zivin, and Neilson (2016) study the effect of fetal exposure to pollution in Chile between 1990 and 2005. The levels of pollution in Chile dropped by more than fifty percent during the study period. The authors take a novel approach to the problem of geographic sorting, focusing on within-family fixed effects. They find that a one standard deviation increase in carbon monoxide exposure in the third trimester reduces language and mathematics scores by 0.042 and 0.038 standard deviations in the fourth grade.

2.4 *Other early exposures*

This paper is part of a larger literature on the effects of adverse environmental shocks. The focus of this paper is pollutants – such as the U.S. criteria pollutants – that are ordinarily present in the urban environment, but there is growing evidence that other kinds of man-made and natural environmental hazards can have a profound effect on health. Currie and Schmeider (2009), Agarwal et al (2010) and Persico et al (2016)

[8]Bharadwaj, Graff Ziven, Mullins, and Neidel (2016) study the effect of early exposure to the Great London Smog on the development of asthma using data from the English Longitudinal Study of Aging. The data do not allow a large sample size - 42 people in the data were exposed in utero, and 15 at less than one year old - but the estimated coefficients map well with the pattern of Smogs in London, with higher reports of Asthma among London cohorts affected by the 1948 and 1952 Smogs.

[9]Evidence on other harmful exposures, such as from radioactive fallout or toxic waste, is discussed later in this section.

study the effects of toxic releases. There is also growing evidence about exposure to water pollution (Gamber-Rabindran et al 2010), exposure to radiation (Almond et al 2009, Black et al 2013) and to dust clouds (Baek et al 2015, Arthi et al 2016).

This study is also related to a broader literature on the effects of *in utero* shocks. The earliest contribution in the economics literature is by Almond (2006), who studied the effect of fetal exposure to the the 1918 influenza epidemic. The later literature – see Almond & Currie (2011) for a review – has shown lasting effects from fetal exposure to poor health conditions (Bleakley 2007, Field et al 2009, Lin & Liu 2014, Barreca 2010, Kelly 2011) alcohol (Nilsson et al 2008), poor dietary conditions (Almond et al 2015, Chen & Zhou 2007), economic conditions (Wehby 2016, Burlando 2014), stress or bad news (Carlson 2014, Aizer et al 2015,), warfare (Lee 2014), or weather shocks (Groppo and Kraehnert 2015, Maccini & Yang 2009).

3 The Great London Smog of 1952

This section provides information about the Great London Smog, including how it occurred, reaction and reportage of the event, environmental conditions at the time, and effects on mortality.

The Great London Smog started on the fifth of December, 1952, and lasted five days in total. It was a caused by an atmospheric temperature that accompanied a high pressure weather system that had settled over London. Pollution from traffic and the burning of coal became trapped under an envelope of cold air, and a thick ground-level smog formed over the city[10]. Conditions persisted for four more days, during which time visibility dropped, and pollution levels increased threefold. Londoners were accustomed to thick winter fogs - the most recent smog had been in 1948 - and there was little panic. Reports at the time indicate that people stayed at home[11] or carried on with ordinary life. The Times of London reported that there were traffic problems as a result of reduced visibility. There were also reports about the cancellation of sporting fixtures, and an increase in housebreaking, but no reports relating to human health[12]. When official figures on deaths and hospitalisations arrived a week later, it became clear that there had been a dramatic effect on the population. A Ministry of Health (1954) report attributed four thousand premature deaths – mainly among the over sixty fives (see Figure 8) – to these five days of exposure[13]. Parliament responded with Clean Air Act of 1956, drafted with the goal of preventing any further smogs in London.

[10] Temperature inversions of the kind that affected London in 1952 are a common occurrence in many cities worldwide. Examples of cities in which inversions are particularly frequent are Los Angeles, Mexico City, Mumbai, Chengdu, Santiago, Sao Paolo, Tehran, and Milan.

[11] Houses in London were not (and are not) well sealed against the outside air.

[12] A report in the Times of December the 8th noted that some show cattle being transported to the Smithsfields show had encountered breathing difficulties.

[13] The four thousand excess deaths recorded in the next three months were initially attributed to influenza - but there was no evidence of influenza in the lungs of the diseased (Ministry of Health, 1954) and the Chief Medical Officer concluded that there was no major outbreak of influenza in 1952. Later studies have revised the number of excess deaths caused by the smog up to 12,000 Bell & Davis (2001). For three recent papers studying the link between pollution and mortality in London using historical data, see Hanlon & Tian (2015), Beach & Hanlon (2018), and Hanlon (2018).

Historic pollution data for the UK goes back to around the mid-Forties, and it is possible to observe the Smog in context. Figure 3 shows black smoke particulates in London and urban areas of the UK from the period from 1950 to 1958. Annual averages are high in both series - at just over 200 $\mu g/m^3$ BS overall, and closer to 300 $\mu g/m^3$ BS during winter months[14]. In general, seasonal and annual dynamics are similar. The London smog can be seen as a spike in December 1952, and a smaller London smog can also be seen in 1955. Variation in weather conditions is not large in the UK compared to other countries, either over time or across space. Figure 3 shows minimum temperatures in London and for the rest of England. Minimum temperatures in the fifties were low, relative to long-run trends, but there was a not a lot of variation between years.

The Smog impact was well focused on London - Figures 4, 5 and 6 show daily measurements taken during the first half of December for London, Great Britain, and for other large towns. Meters outside of London show a very small increase in pollution levels on the fourth and fifth of December, but nothing close to the scale of the London Smog[15]. Exposure to pollution during the Smog cut across social lines. The most severely affected areas were both affluent (Kensington, Chelsea) and relatively deprived (South London). Figure 1 shows the areas of London where the Smog was most severe. The Figure is based on a report by The Ministry of Health (Wilkins, 1954) that used information from 117 sulphur dioxide meters to divide London into four approximate levels of impact. (The meter level information on which the map was based were sadly not kept) A Londoner in the five days of the smog experienced an average 'spike' in pollution exposure of over 1,100 $\mu g/m^3$ BS. This is a very severe pollution shock - closer in magnitude to serious pollution events in seen Mumbai and Beijing in recent years than to exposures seen in Europe or in the U.S[16].

The effect on mortality can be seen in the registrar data. Figure 7 shows the ratio of deaths in London to those in England and Wales. The impact of the smog is clearly visible, and there are no comparable incidents in the ten year period that the data

[14] Conversion between Black Smoke particulates and TSP is imprecise and depends on local conditions. Bell and Davis (2001) also study the Great London Smog and convert Black Smoke particulates directly into TSP. The conversion rate during the fifties was likely close to 1:1 in winter months. The ratio changes in the summer, and TSP readings for annual averages are likely to be twenty percent higher than those for black smoke. Ball & Hume (1977) investigate traffic pollution in the 1970s in London using both Black Smoke and TSP meters, finding a winter conversion of around 1.14. However, the study is from twenty years later than the Smog, when black smoke stains were less pronounced. Commins & Waller (1966) also study particulates in London, using Black Smoke and TSP meters, for the period from 1955 to 1962, with results that imply a conversion factor of 1.25 for an annual average (and a lower number for readings taken during the winter.)

[15] A notable exception is Leeds (not included in Figure 6, or in the main analysis), which experienced peaks in pollution exposure comparable to those in London.

[16] Kernel density plots based on readings from the Town Hall in Mumbai (from the Indian Central Control Board) suggests that daily average readings of over 800 $\mu g/m^3$ TSP are likely to be observed almost every year in central Mumbai, and readings of over 1000 $\mu g/m^3$ TSP almost every other year. Data from the U.S. Embassy in Beijing suggests that daily average readings of over 800 $\mu g/m^3$ TSP are likely to be observed every other year in Beijing, and readings of 1000 $\mu g/m^3$ TSP around once every five years. Data on cities are rarely available at daily frequencies for low- and middle-income countries, and are particularly difficult to obtain for cities with high levels of pollution, where the incentives to collect and distribute data can be weak. Data collected by Greenstone & Hanna (2014) for India between 1987 and 2007 shows that residential areas of India have experienced daily maximums of over $3000 \mu g/m^3$ TSP - though pollution spikes of this size are very rare.

covers. Deaths were concentrated among the over-forty fives. Figure 8 shows the total number of deaths recorded during the weeks following the smog, broken down by age.

4 Sources of Data

Once the risk of infant mortality has passed, the most serious effects of exposure to atmospheric pollution could take several decades to manifest. The main focus of this paper is to follow those exposed to the Great London Smog as infants or *in utero*, and gather evidence on their health and labour market outcomes over the next sixty years. This section outlines the data sources for long-term outcomes, key independent variables, and on birth outcomes used in the subsequent analysis.

4.1 Long-term labour market and health

Information on long-term outcomes comes from the Office of National Statistics Longitudinal Study. This is a study based on a 1% sample of the decennial census for England and Wales, with inclusion into the study determined by being born on one of four dates in a year. (The four birthdates used to select members of the ONS-LS are treated as strictly confidential, and are not disclosed to data-users.) Participants are observed in the 1971, 1981, 1991, 2001, and 2011 censuses, and additional information on major events such as births, migrations, cancer registrations, and deaths have been linked by the Office of National Statistics. The full study holds information on just under a million individuals born in England and Wales. For the purpose of this paper, individuals who were *in utero* in London during the Smog are identified by their year and borough[17] of birth. The full dataset used in this paper contains information from around 42,000 people born between 1950 and 1958, and observed in each round of the census. Around 6,600 of these people were born in London, with an average of 730 individuals born in any one year. Summary statistics can be seen in Table 1. As discussed in Bertrand *et al* (2004), performing the analysis with individual-level data could lead to inconsistent standard errors if there existed a random effect at the area-year level. Following the authors, the dataset is collapsed into area-birthyear cells, resulting in a balanced panel with fifty one geographic areas observed from 1950 to 1958.

4.2 Exposure to pollution and extreme weather

Both severe weather and pollution are expected to affect registered births in a given quarter. Data on weather conditions comes from the U.K. Meteorological Office, and includes monthly series for London and the rest of England and Wales for minimum temperatures, maximum temperatures, days of frost, and precipitation. A graph showing the temperature series for London and the UK can be seen in Figure 3. Seasonal

[17] Greater London is divided into 32 Boroughs, which average around 40 square kilometres in size, and had average populations of around 100,000 at the time of the smog. In the E.U. categorisation, one or two Boroughs form a NUTS3 region.

changes tend to affect different parts of the U.K. symmetrically. Winter temperatures in London during 1952 were low relative to long-term trends, but almost identical to those in the five year period from 1951 to 1955. Data on black smoke particulates comes from the official publications of the Fuel Research Board. There were around two hundred black smoke meters active in England and Wales during the period, though not all meters can be used to construct long series. Figure 3 shows a series for urban areas of England and Wales based on measurements from fifty eight meters, and a series for London, based on data from fifteen meters. In general, seasonal and annual dynamics are similar in different parts of England and Wales.

4.3 Evidence on fetal loss

Before proceeding to the main analysis, the paper will look at the extent to which exposure to the Great London Smog affected fetal loss. Information on births outcomes is based on historic data from the Registrar General for Births, Deaths, and Marriages. This is the official record of births for the United Kingdom, and should contain the universe of registered births[18]. Two datasets have been constructed from the information available. The first is a quarterly time-series for births in London and the rest of England and Wales spanning from 1947 to 1964, which makes it possible to observe the Great London Smog in context. Figure 10 gives a long-view of births over this period, while Figure 11 shows the ratio of the two series. Overall, London and the rest of England and Wales share seasonal and broad long-term trends. There were two serious pollution events reported during the period. The first was a London smog in the winter of 1948, and the second was the Great London Smog in the winter of 1952. Both appear to register in the ratio series - with a dip in births coincident with when first trimester births exposed in 1948 would have been registered, and a long dip in 1953, highlighted in gray. (Those who were *in utero* in London during the Smog in December of 1952 would be born in 1953.) The second dataset is a quarterly panel broken down to the Borough[19] level. This data spans from the first quarter of 1947 till the fourth quarter of 1954. Summary statistics from this panel are presented in the following section.

5 The Effect of the Smog on Fetal Loss and Infant Mortality

Although the Smog lasted just a few days, the impact on fetal loss would be observed for up to nine months. This is because mothers were exposed at different stages in their pregnancies: fetal loss from exposure in the first trimester would be observed six to nine months later, while the effects of exposure in the third trimester would be observed soon after December. As discussed in earlier sections, early exposure to pollution is likely

[18] Registration of births has been compulsory in the U.K. since the Births and Deaths Act of 1874.

[19] Greater London is divided into 32 Boroughs, which average around 40 square kilometres in size, and had average populations of around 100,000 at the time of the smog. In the E.U. categorisation, one or two Boroughs form a NUTS3 region.

to be particularly serious, as this is when all critical systems are formed. A loss of the foetus at this stage would not be included in stillbirth records[20], and could potentially go unnoticed by the mother. Following from Jayachandran (2009), the approach of this section is to study the effect of the smog by observing 'missing' births in subsequent months. This measure is a good proxy for fetal loss, and has the advantage of being able to capture stillbirths, un-reported stillbirths, and fetal losses happening earlier in the pregnancy,

Data on births comes from the Registrar General for England and Wales, and takes the form of a quarterly panel broken down to the Borough[21] level. This data spans from the first quarter of 1947 till the fourth quarter of 1954. Summary statistics for the dependent variable and key controls are shown in Table 1[22]. The dependent variable is births per one thousand people B_{jt} in borough j, and quarter t. It is modelled as a function of environmental exposures E_{jt}, a vector containing levels and squares of in-utero minimum temperatures, and average pollution exposure when in utero. Births show a clear seasonality, and four dummies $\sum_{i=1}^{4} s_{it}$ are included in the model controls for season of birth. In order to control for unobserved nation-wide shocks, a differences-in-differences strategy is used. Below, D_j^L is a dummy for boroughs in London exposed the smog, and D_t^{53} is a dummy for the year 1953, when births affected by the December 1952 smog would be registered. Some specifications also include borough-level fixed effects λ_j, and year-level fixed effects μ_t.

$$B_{jt} = \alpha + \beta_1 D_j^L + \beta_2 D_t^{53} + \beta_3 (D_j^L D_t^{53}) + \beta_4 E_{jt} + \sum_{i=1}^{4} s_{it} + \lambda_j + \mu_t + \epsilon_{jt} \tag{1}$$

The coefficient of interest is β_3, which can be interpreted as the change in births per one thousand people as a result of exposure to the Great London Smog. A key identifying assumption is that the effects of the Great London Smog did not extend out to the rest of England and Wales. Daily exposures to pollution during the Smog can be seen in Figures 5 and 6. Pollution levels in the rest of the UK and in other big towns do not show evidence of increased intensity during the period of the smog. When taking account of unobserved nation-level effects, a second key assumption is that London and the rest of England and Wales follow common trends in 1953. Trends relative to the UK were estimated using data from years unaffected by the Great London Smog. It was not possible to reject the null hypothesis that births-per-capita in London and the UK followed a common trend. The statistical model is estimated by ordinary least squares, with robust standard errors clustered at the borough-level. Results for the key parameters can be seen in Table 4. The estimated coefficient for β_3 was -0.09 for all models. The population of the affected Boroughs was around 4,750,000 in the fourth

[20] A stillbirth is defined as a fetal loss after 24 weeks of pregnancy.

[21] Greater London is divided into 32 Boroughs, which average around 40 square kilometres in size, and had average populations of around 100,000 at the time of the smog. In the E.U. categorisation, one or two Boroughs form a NUTS3 region.

[22] Figures for the control variables vary from the summary statistics presented in the data section because the number of years covered is smaller.

quarter of 1952. The estimated reduction in births per thousand people implies that there were around 1280 fewer births in London in the three quarters following the Great London Smog. This represents around a 2% reduction in the size of the cohort *in utero* in London during the Smog.

There is a significant body of evidence linking early exposure to pollution with infant mortality. In order to investigate this possibility, the exercise was repeated with deaths among infants less than one year old per 1000 people as the dependent variable. Unlike the analysis on missing births, it is not possible to distinguish cleanly between the effects of *in utero* and infant exposure when looking at infant deaths with quarterly data. This is because a child born in a given quarter could potentially appear in the infant mortality statistics for four successive quarters[23]. Results can be seen in the Table 5. The variable 'infant deaths after the Smog' measures the increase in deaths among infants less than one year old, per 1000 people, in the year following the Smog. The variable 'Infant deaths in quarter of the Smog' shows the number of deaths among infants born in the final quarter of 1952. This group is treated separately because only around a fifth would have been in-utero during the smog. Results for both variables show an increase of 0.01 - this is equivalent to an increase of fifty infant deaths per quarter, or a total of around 250. The number of infant deaths per 1000 people was around 0.135 per quarter in previous years, and so the percentage change in infant deaths is around 7%. Although a large percentage increase in mortality, the effect on the size of the surviving cohort is small, at around 0.2%.

6 Long-term effects for survivors

The most serious effects of pollution exposure can take decades to manifest. While the effects of education and employment can be observed after twenty or thirty years, an increased chance of developing cancer or cardiovascular problems would usually be observed from the late-forties onwards. The goal of this section is to follow cohorts of people who were infants or *in utero* in London during the Great Smog, and observe long-term effects on educational outcomes, employment through the life-cycle, and vulnerability to serious risks among the surviving population.

Data on long-term outcomes comes from a balanced area-birthyear panel of labour market and health outcomes, which covers cohorts born in 1950 through to 1958, and is based on the Office of National Statistics Longitudinal Study[24]. The outcome variable Y_{jt} is derived from information on individual labour market and health outcomes collapsed to the area j and birth-year t level. The variable D_t^{IU} is a dummy that takes the value of one if cohort jt was *in utero* during the Smog, and the variable D_t^{IN} is a dummy taking the value of one if the cohort were less than one year old during the Smog. These are interacted with D_j^{Lon}, a dummy that takes the value one if cohort jt

[23] Thus, 1952q4 statistics contain information from only those who were *in utero* during the Smog, 1953q4 contains information from those who were infants during the Smog, and quarters in-between contain a mix of the two.

[24] For more detail on data used and summary statistics, see Section 4.

were born in an area of London. $\mathbf{E_{jt}}$ contains levels and squares of in-utero minimum temperature and pollution exposure. $\mathbf{X_{jt}}$ contains collapsed individual information on gender and ethnicity. ζ_j and η_t are area and year fixed effects. τ_j contains separate linear time trends for areas in London and in the rest of England and Wales.

$$Y_{jt} = \alpha + \beta_1(D_t^{IU}.D_j^{Lon}) + \beta_2(D_t^{IN}.D_j^{Lon}) + \mathbf{E_{jt}}\boldsymbol{\gamma} + \mathbf{X_{jt}}\boldsymbol{\delta} + \zeta_j + \eta_t + \tau_j t + \epsilon_{jt} \quad (2)$$

The coefficients of interest are β_1 and β_2. They can be interpreted as the difference in long-term outcomes for cohorts who were *in utero* or infants in London during the smog, compared to other London cohorts, taking into account long-run average levels of pollution exposure, possible national-level shocks and long-run trends.

Some important effects of the Smog would not be captured by this specification. All birth cohorts in London would have been affected by the Smog to some extent - for example, the Smog caused adult mortality, concentrated among the elderly, which could affect inheritances and the availability of childcare. This would not come through in β_1 and β_2 because the comparison is between London cohorts. Similarly, all birth cohorts in London would also have been affected by high average levels of pollution. As average exposures to severe weather and pollution are controlled for directly, with year-level dummies, and with linear trends, these long-run exposures would not be captured by β_1 or β_2. Differences in long-run outcomes for cohorts reflected in β_1 and β_2 are expected to be driven by poor health, but to be mitigated by potential parental responses. For example, a parent might take extra care of a sickly child. Lastly, the parameters β_1 and β_2 should capture effects at the cohort level, rather than the individual level. As a consequence, a particularly severe shock to health could increase the average health in the cohort by removing the most vulnerable through infant mortality or fetal loss. Evidence on fetal loss from the previous section suggests that around 2% of the treated population did not survive till birth, which would be expected to create a shift towards healthier outcomes in β_2 for the affected cohort. As in all studies of long-term outcomes from early shocks, estimates must therefore be treated as causal effects for the cohort, but lower bounds for individuals.

An important assumption in in identifying β_1 and β_2 is that other urban areas of England and Wales[25] were unaffected by the 1952 Smog. Daily exposures to pollution during the Smog can be seen in Figures 5 and 6. Pollution levels in the rest of the UK and in other big towns do not show evidence of increased intensity during the period of the Smog. A second, implicit, assumption is that the model is correctly specified, and that the set of controls, fixed effects, and trends sufficiently isolate the effects of being *in utero* or an infants in London during the Smog, rather than some other cohort. Results will be reported that omit environmental and demographic controls, and that omit linear time trends. Discussion of estimates from the model will focus on the full model, shown always in Column (4). Estimation is by ordinary least squares, with robust standard

[25] Rural areas were defined as those with population density less than 400 people per square kilometer, and removed from the sample.

errors clustered at the area-level.

6.1 Outcomes Characterising the Surviving Population

There is evidence from the historic registrar data that exposure to the Great London Smog had a significant effect on the size of the surviving population. A natural first step is to gather evidence on the surviving cohort. There are two outcomes in the data, gender and ethnicity, that would be fixed at conception, making it possible to gather evidence on the effect of the Smog on the composition of the surviving population.

As discussed in the literature review, the hypothesis that females are more likely to survive shocks has found empirical support in both the epidemiological and economics literatures. Sanders and Stoecker (2011), for example, studied the effects of pollution exposure on the gender of survivors, finding that a standard deviation increase in pollution exposure decreased the percentage of births that are male by 3.1%. Estimates from the Equation 2 can be seen in Table 6. A graph of gender in London and urban areas of England and Wales can be seen in Figure 12. The results from the full model are in column (3). (Models 2 and 4, which would include gender as a control were not estimated, because gender is the outcome variable.) The cohort who were *in utero* in London during the Smog are 3% less likely to be male, but the difference is not statistically significant. Those who were infants in London during the smog are 5% less likely to be male.

The second characteristic fixed at conception is ethnicity. Results can be seen in Table 7. Overall trends in England and Wales and London diverged over the sample period, as seen in Figure 13. Estimates from the specification in column (3), which allows for differential long-run trends show no effect on the ethnic make-up of the cohort who were *in utero* in London during the Smog. Those who were infants during the smog are 2% less likely to be white, significant to the 5% level.

6.2 Outcomes determined after conception

Educational attainment can capture the effects of weaker health problems that might not appear in hospital records and mortality statistics. Individuals are recorded as degree-holders if they report having a degree in any of the censuses from 1971 through to 2001. The series for London and for urban areas of England and Wales can be seen in Figure 14. The series appear to follow each other closely, except for the cohorts who were *in utero* or infants in London during the smog. Estimated coefficients can be seen in Table 8. Those who were *in utero* in London during the smog are 3% less likely to hold a degree, significant at the 5% level. The estimate for those who were infants in London during the Smog indicated a cohort that was 2% less likely to hold a degree, but this estimate was not significantly different from zero.

Fetal exposure to pollution could affect employment directly through its effects on health, or indirectly through its effects on educational attainment. As with education, changes in employment can be a good measure of the kind of health effects that would not appear in statistics on hospitalisations or mortality. The studied cohorts entered the labour market under very similar conditions. Figure 9 shows that average levels of unemployment on entry were 2.8% while those for the 1953 cohort were 3.1%, and so any employment effects are unlikely to be driven by issues of timing. Individuals are first observed in 1971, when the two cohorts of interest were around twenty and may still have been at university. These cohorts are then observed at four points during the normal working life: in 1981 when thirty, in 1991 when forty, and in 2001 when fifty. Results be seen in Table 9. Overall, there was no significant difference in unemployment for the *in utero* cohorts. The results for the infant cohort are similar, except for in 1991 (when the cohort were around forty years old.) In this year, the cohort who were infants during the smog appear 2% less likely to be unemployed.

Hours worked were observed in 1971, 1991 and 2001, when the cohorts who were infants or *in utero* in London during the Smog were around twenty, forty, and fifty years old. Graphs of the three series can be seen in Figures 15, 16, and 17. The two series follow each other closely in other years. Working hours shown in the graph for the 1971 census tail off for birth years later than 1956 as many are too young to work legally. (The working age in the U.K. is sixteen.) Results can be seen in Table 10. The cohort who were *in utero* in London during the Smog work eight fewer hours a week than their peers in 1971, and just over an hour less a week in 1991, though the second estimate is significant only at the 10% level when including controls for gender and ethnicity. There is no significant difference by 2001. The cohort who were infants in London during the Smog work four fewer hours a week in 1971, though this estimate is significant only at 10% level. The estimate for 1991 is around negative one, but not significant, and there is no difference in hours by 2001.

Fetal loss or infant mortality as a result of early exposure ot the Great London Smog would tend to be concentrated among those most likely to become unhealthy adults. In the data, this would be equivalent to truncating the left-hand tail of the health distribution. The employment data in the ONS Longitudinal Study includes a question that could shed light on whether there are fewer adults with very poor health in the sample. Respondents are asked to answer the following question: 'I am permanently out of employment for health reasons.' This question was asked in a consistent way over the respondents' life-cycle and, being a more concrete measure than self-reported health, avoids some conceptual issues with that measure[26]. Results can be seen in Table 11. The cohort that was *in utero* during the Smog was simlilar to other cohorts in 1971, 1981, and 1991. In the first two decades, the estimate was negative and significant, but very small. They were 2% less likely to be permanently sick in 2001, and 1% less likely in 2011, though this latter result is not significantly different from zero. For those who

[26]Deaton (2008) discusses three key issues. The first is that people might not fully perceive the impacts of a health shock. Someone with small respiratory problems may not fully contemplate the career as a professional footballer they might have had in full health. The second is that people grow accustomed to their ailments, and no longer consider them to be day-to-day problems. The third is that there are cross-country differences in how this kind of question is answered due to both cultural differences, and differences in the average health of comparison groups.

were infants in London during the Smog, there are no significant differences in severe illness.

Exposure to atmospheric pollution has been linked to cancer in numerous studies, but there has been no evidence to date on the effects of fetal or infant exposures, because it can take many years for cancers to develop. A graph of cancer incidence in London and urban areas of England and Wales can be seen in Figure 18. Results can be seen in Table 12. For the cohort that were *in utero* during the smog, there was no effect on cancer registration. The cohort who were infants in London during the Smog (and so directly exposed) were 3% more likely to develop a cancer.

The final outcome observed is mortality, defined as dying before 2011, when the 1952 and 1953 cohorts would be around sixty years old. A graph of mortality series for London and urban areas of England and Wales can be seen in Figure 19. Results can be seen in Table 19. They show that cohorts who were *in utero* in London during the Smog who were infants are both 2% less likely to have died by 2011. The section on missing births showed around a 2% reduction in the *in utero* cohort due to fetal loss, and evidence on gender within the infant cohort also suggests an effect on the size of the surviving cohort. Taken together, it may be that those not surviving to be observed are less healthy than the average in the cohort, and would have been more likely to die before sixty.

7 Discussion

The goal of this study was to investigate the long-term effects of early exposure to the Great London Smog. The results showed that the Smog had two serious effects on the affected *in utero* and infant cohorts: early loss among the most vulnerable, and lasting damage to the surviving population.

Early loss among the most vulnerable The first effect of the Smog was an increase in fetal and infant mortality. Evidence from the Registrar General showed around 1280 fewer births in the year following the Smog than would be expected, or a reduction of 2% in the size of the *in utero* cohort. Infant deaths were also 7% higher following the smog, though it was not possible to determine if this increase was among those affected *in utero* or as infants. There is evidence in the epidemiological and economic literatures suggesting that males are more vulnerable to *in utero* health shocks[27]. The surviving population were first observed in 1971 at around twenty years old. For the cohort who were *in utero* in London during the Smog, there was no significant evidence that fetal loss was concentrated among males. The cohort who were infants in London during the Smog were 5% less likely to be male, indicating a considerable difference in survival between male and female cohorts exposed to the Smog as infants.

Fetal or infant mortality would also be expected to be concentrated among the most vulnerable. Evidence from two outcomes, both associated with serious poor health,

[27]See, for example, Sanders and Stoecker (2011), and the papers cited within,

provided support for this hypothesis. Those exposed *in utero* were significantly less likely to be 'permanently out of work for reasons of poor health' than their peers, though the difference was small, peaking at 2% at around the age of fifty, and not always statistically significant. Stronger, was the evidence on dying before the age of around sixty. The cohorts who were *in utero* or infants in London during the Smog were both 2% less likely to have died young, suggesting that those not surviving fetal or infant exposure would have been more likely to die young, in the absence of the smog[28]. The observed effects were robust to controls for the proportion of men and ethnic minorities in the cohort, and environmental controls including levels and squares of temperature and black smoke particulate pollution.

Lasting damage to the surviving population Evidence on other outcomes indicates that exposure to the five day smog created lasting damage to health that affected labour market outcomes, human capital accumulation, and health. The strongest differences were in the twenties and thirties, the years in which labour market trajectories are largely determined. In 1971, those who were *in utero* in London during the Smog worked eight hours less than other cohorts, and those who were infants during the Smog worked four fewer hours. This effect faded slowly, either as the members of these cohorts adjusted, or as other London cohorts 'caught up' in terms of poor health. By 1991, when the 'early exposure' cohorts were around forty, those who were *in utero* during the Smog worked between one and two hours less, and there was no significant difference for those who were infants at the time. The evidence was not consistent with a difference in unemployment, but the two richest specifications showed a significant reduction in human capital accumulation, with those who were *in utero* during the smog three percentage points less likely to hold a degree-level qualification. Lastly, estimates from the two richest specifications showed those exposed to the smog directly as infants were 3% more likely to develop a cancer, implying that some of the damage from the smog lay latent for several decades. The observed effects were, again, robust to controls for the proportion of men and ethnic minorities in the cohort, and environmental controls including levels and squares of temperature and black smoke particulate pollution.

Comparison with modern smogs The Great Smog was a particularly severe event, and the last major smog event in London was in 1962. The level of average Black Smoke particulates was 1,500 $\mu g/m^3$, with a peak daily average of 1,720 $\mu g/m^3$ on the 8th of December. In terms of modern measures, these figures correspond very roughly to 500 and 720 $\mu g/m^3$ PM2.5[29]. Despite the gap of sixty years, the duration and particulate exposures in the Great London Smog are reminiscent of severe pollution events seen in

[28] The alternative explantation, that pollution has beneficial effects on those affected ('what doesn't kill you make you stronger'), is not consistent with evidence from the wider medical and epidemiological literature. Pollution always has a deleterious effect on health.

[29] Conversion of Black Smoke measures to PM2.5 is imprecise. Bell *et al* (2004) convert London's 1952 Black Smoke measures to TSP at (1:1). Cao *et al* (2011) convert TSP to PM2.5 at a ratio of (3:1) in the context of the P.R. China.

recent years. Short smogs have been recorded in India and China in recent years in which measures of particulates have reached daily peaks of 600 $\mu g/m^3$ PM2.5 or higher[30][31].

Pollution data are rarely available at daily frequencies for low- and middle-income countries, and are particularly difficult to obtain for cities with high levels of pollution, where the incentives to collect and distribute data can be weak. Evidence from cities that *have* invested in monitoring networks suggests that severe smogs are not rare events. Kernel density plots based on readings from the Town Hall in Mumbai (from the Indian Central Control Board) and the U.S. Embassy in Beijing suggest that daily average readings over 800 $\mu g/m^3$ TSP occur almost every year in central Mumbai, and almost every other year in Beijing. Daily average readings of over 1000 $\mu g/m^3$ TSP are likely to occur almost every other year in central Mumbai, and about one year in five in Beijing. Data collected by Greenstone & Hanna (2014) for India between 1987 and 2007 shows that some residential areas of India have experienced daily peaks of over 3000$\mu g/m^3$ TSP - though pollution readings this high are extremely rare.

Implications for policy The avoidance of high levels of urban pollution is often uneconomical for vulnerable populations who cannot simply move to the countryside for long periods. Urban smogs are brief events, making avoidance both possible and a good investment, if the short- and long-run effects of the Great London Smog are a guide. This would remain the case even in cities with generally high levels of pollution, as was the case in London in the Fifties. The returns to investment in pollution meters and other infrastructure that could be used to warn vulnerable populations could then be high, but these investments are rare. The World Health Organisation was able to collect pollution data from only around 8% of cities - with missing values most common in areas expected to have high levels of atmospheric pollution.

8 Conclusion

This paper estimated the long-run labour market effects of being *in utero* or an infant in London during the Great London Smog. The short-run effects of the Smog on fetal loss were investigated using historical data from the Registrar General for England and Wales. Those *in utero* during the Smog would have been born in 1953. Results showed around 1280 fewer births in that year than would be expected, or a reduction of 2% in the size of the *in utero* cohort. Those observed later in life were 3% less likely to hold a degree-level qualification. There was no effect on unemployment, but there was a significant effect on hours worked in the early career, with those who were *in utero* in London during the Smog working 8 fewer hours a week, and those who were

[30]Guardian, 24th August 2013, "China hit by another airpocalypse as air pollution cancer link confirmed" available at: https://www.theguardian.com/environment/chinas-choice/2013/oct/24/china-airpocalypse-harbin-air-pollution-cancer

[31]Hindustan Times, 11th November 2017,"Air clean-up act: PM10 out of emergency levels, PM2.5 to follow soon" Available at: http://www.hindustantimes.com/delhi-news/air-clean-up-act-pm10-level-out-of-emergency-levels-pm2-5-to-follow-soon/story-uygP86Y7UKhisyI8teiUIL.html

infants during the Smog working 4 fewer hours a week. There was no effect on cancer registration for those who were *in utero* in London during the Smog, but those who were infants (and so exposed to the Smog directly) were 3% more likely to be diagnosed with cancer. Lastly, the treated cohorts were both 2% less likely to die in sample - suggesting that those not surviving to be observed would have died before the age of sixty, in the absence of the Smog. The results of this paper imply that the costs exposure to the Great London Smog persisted long into adulthood for those who were affected young. The Great Smog was over sixty years ago, but severe urban smogs remain. The 2013 smog in North-East China lasted five days and saw hourly pollution levels of over 1,000 $\mu g/m^3$ PM2.5 in Harbin, a city of more than five million people. If the long-term costs of the London Smog are any guide, then increased infrastructure to allow vulnerable populations to avoid such events could be a good long-term investment.

References

Agarwal, N., C. Banternghansa, and L. Bui (2010) "Toxic Exposure in America: Estimating Fetal and Infant Health Outcomes from 14 Years of TRI Reporting," *Journal of Health Economics*, 29, pp. 557–574.

Aizer, A., L. Stroud, and S. Buka (2016) "Maternal Stress and Child Outcomes: Evidence from Siblings," *Journal of Human Resources*, 51, No. 3, pp. 523–555.

Almond, D. (2006) "Is the 1918 Influenza Pandemic Over? Long-Term Effects of In Utero Influenza Exposure in the Post-1940 U.S. Population," *Journal of Political Economy*, 114, No. 4, pp. 672–712.

Almond, D. and J. Currie (2011) "Killing Me Softly: The Fetal Origins Hypothesis," *Journal of Economic Perspectives*, 25, No. 3, pp. 153–172.

Almond, D., L. Edlund, and M. Palme (2009) "Chernobyls Subclinical Legacy: Prenatal Exposure to Radioactive Fallout and School Outcomes in Sweden," *Quarterly Journal of Economics*, 114, No. 4, pp. 1729–1772.

Almond, D. and B. Mazumder (2013) "Fetal Origins and Parental Responses," *SSRN Working Paper*, 2012-14.

Almond, D., B. Mazumder, and R. van Ewijk (2015) "In Utero Ramadan Exposure and Children's Academic Performance," *Economic Journal*, 125, No. 589, pp. 1501–1533.

Arceo, E., R. Hanna, and P. Oliva (2016) "Does the Effect of Pollution on Infant Mortality Differ Between Developing and Developed Countries? Evidence from Mexico City," *The Economic Journal*, 126, No. 591, pp. 257–280.

Arthi, V. (2016) ""The Dust Was Long in Settling": Human Capital and the Lasting Impact of the American Dust Bowl.," *Oxford Economic and and Social History Working Papers*, 129.

Baek, D., D. Altindag, and N. Mocan (2015) "Chinese Yellow Dust and Korean Infant Health," *NBER Working Paper*, 21613.

Ball, D. and R. Hume (1977) "The Relative Importance of Vehicular and Domestic Emissions of Dark Smoke in Greater London in the Mid-1970s, the Significance of Smoke Shade Measurements, and an Explanation of the Relationship of Smoke Shade to Gravimetric Measurements of Particulate," *Atmospheric Environment*, 11, pp. 1065–1073.

Barreca, A. (2010) "The Long-Term Economic Impact of In Utero and Postnatal Exposure to Malaria," *Journal of Human Resources*, 45, No. 4, pp. 865–892.

Beach, B. and W. W. Hanlon (2018) "Coal Smoke and Mortality in an Early Industrial Economy," *Economic Journal*, 128, No. 618, pp. 2652–2675.

Bell, M., D. Davis, and T. Fletcher (2004) "A Retrospective Assessment of Mortality from the London Smog Episode of 1952: The Role of Influenza and Pollution," *Environmental Health Perspectives*, 112, pp. 6–8.

Bell, M. and D. Davis (2001) "Reassessment of the Lethal London Fog of 1952: Novel Indicators of Acute and Chronic Indicators of Acute Exposure to Air Pollution," *Environmental Health Perspectives*, 109, No. 3, pp. 389–394.

Bertrand, M., E. Duflo, and S. Mullainathan (2004) "How Much Should We Trust Differences-in-Differences Estimates?" *Quarterly Journal of Economics*, 119, No. 1, pp. 249–275.

Bharadwaj, Gibson, J. Graff Zivin, and Neilson (2014) "Gray Matters: Fetal Pollution Exposure and Human Capital Formation," *NBER Working Paper*, 20662.

Bharadwaj, P., J. G. Zivin, J. T. Mullins, and M. Neidell (2016) "Early-Life Exposure to the Great Smog of 1952 and the Development of Asthma," *American Journal of Respiratory and Critical Care Medicine*, 194, No. 12, pp. 1475–1482.

Black, S., A. Bütikofer, P. Devereux, and K. Salvanes (2014) "This Is Only a Test? Long-Run Impacts of Prenatal Exposure to Radioactive Fallout from Nuclear Weapon Testing," *NBER Working Paper*, 18987.

Bleakley, H. (2007) "Disease and Development: Evidence from Hookworm Eradication in the American South," *Quarterly Journal of Economics*, 122, No. 1, pp. 73–117.

Bozzoli, C., A. Deaton, and C. Quintana-Domeque (2009) "Child Mortality, Income, and Adult Height," *Demography*, 46, No. 4, pp. 647–669.

Burlando, A. (2014) "Transitory Shocks and Birth Weights: Evidence from a Blackout in Zanzibar," *Journal of Development Economics*, 108, pp. 154–168.

Carlson, K. (2014) "Fear Itself: The Effects of Distressing Economic News on Birth Outcomes," *Journal of Health Economics*, 41, pp. 117–132.

Chay, K. Y. and M. Greenstone (2005) "Does Air Quality Matter? Evidence from the Housing Market," *Journal of Political Economy*, 113, No. 2, pp. 376–424.

Chay, K. and M. Greenstone (2003) "The Impact of Air Pollution on Infant Mortality: Variation in Pollution Shocks Induced by a Recession," *Quarterly Journal of Economics*, 118, No. 3, pp. 1121–1167.

Chen, Y. and L.-A. Zhou (2007) "The Long-Term Health and Economic Consequences of the 1959 - 1961 Famine in China," *Journal of Health Economics*, 26, pp. 659–681.

Cho, S., C.-K. Lee, and B. Kim (2013) "The Impacts of Air Pollution on Low Birth Weight," *Applied Economics Letters*, 20, pp. 208–212.

Clay, K., J. Lewis, and E. Severnini (2016) "Canary in a Coal Mine: Infant Mortality, Property Values, and Tradeoffs Associated with Mid-20th Century Air Pollution," *NBER Working Paper*, 22155.

Commins, B. and R. Waller (1966) "Observations from a Ten-Year-Study of Pollution at a Site in the City of London," *Atmospheric Environment*, 1, pp. 49–68.

Currie, J., L. Davis, M. Greenstone, and R. Walker (2015) "Environmental Health Risks and Housing Values: Evidence from 1,600 Toxic Plant Openings and Closings," *American Economic Review*, 105, No. 2, pp. 678–709.

Currie, J. and M. Neidell (2005) "Air Pollution and Infant Health: What Can We Learn from California's Recent Experience?" *Quarterly Journal of Economics*, 120, No. 3, pp. 1003–1030.

Currie, J. and J. F. Schmieder (2009) "Fetal Exposures to Toxic Releases and Infant Health," *American Economic Review*, 99, No. 2, pp. 177–183.

Currie, J. and T. Vogl (2013) "Early-Life Health and Adult Circumstance in Developing Countries," *Annual Review of Economics*, 5, No. 1, pp. 1–36.

Currie, J. and R. Walker (2011) "Traffic Congestion and Infant Health: Evidence from E-ZPass," *American Economic Journal: Applied Economics*, 3, No. 1, pp. 65–90.

Field, E., O. Robles, and M. Torero (2009) "Iodine Deficiency and Schooling Attainment in Tanzania," *American Economic Journal: Applied Economics*, 1, No. 4, pp. 140–169.

Gamper-Rabindran, S., S. Khan, and C. Timmins (2010) "The Impact of Piped Water Provision on Infant Mortality in Brazil: A Quantile Panel Data Approach," *Journal of Development Economics*, 92, pp. 188–200.

Graff Zivin, J. and M. Neidell (2013) "Environment, Health, and Human Capital," *Journal of Economic Literature*, 51, No. 3, pp. 689–730.

Greenstone, M. and R. Hanna (2014) “Environmental Regulations, Air and Water Pollution, and Infant Mortality in India,” *American Economic Review*, 104, No. 10, pp. 3038–3072.

Groppo, V. and K. Kraehnert (2015) “The Impact of Extreme Weather Events on Education,” *DIW Working Paper*, 1534.

Isen, A., M. Rossin-Slater, and R. Walker (2017) “Every Breath You Take - Every Dollar You’ll Make: The Long-Term Consequences of the Clean Air Act of 1970,” *Journal of Political Economy*, (forthcoming).

Jayachandran, S. (2009) “Air Quality and Early-Life Mortality Evidence from Indonesia’s Wildfires,” *Journal of Human Resources*, 44, No. 4, pp. 916–954.

Kannan, S., D. P. Misra, J. T. Dvonch, and A. Krishnakumar (2006) “Exposures to Airborne Particulate Matter and Adverse Perinatal Outcomes: A Biologically Plausible Mechanistic Framework for Exploring Potential Effect Modification by Nutrition,” *Environmental Health Perspectives*, 114, No. 11, pp. 1636–1642.

Katharina, J. (2014) “Air Pollution, Avoidance Behaviour and Children’s Respiratory Health: Evidence from England,” *Journal of Health Economics*, 23, pp. 23–42.

Kelly, E. (2011) “The Scourge of Asian Flu In Utero Exposure to Pandemic Influenza and the Development of a Cohort of British Children,” *Journal of Human Resources*, 46, No. 4, pp. 669–694.

Kelly, F. J. (2003) “Oxidative Stress and Its Role in Air Pollution and Adverse Effects,” *Occup Environ Med*, 60, pp. 612–616.

Khawand, C. (2015) “Air Quality, Mortality, and Perinatal Health: Causal Evidence from Wildfires,” *Working Paper (2015) available at https://ideas.repec.org/jmp/2015/pkh318.pdf*.

Knittel, C., D. Miller, and N. Sanders (2016) “Caution, Drivers! Children Present: Traffic, Pollution, and Infant Health,” *Review of Economics and Statistics*, 98, No. 2, pp. 350–366.

Lee, C. (2014) “In Utero Exposure to the Korean War and Its Long-Term Effects on Socioeconomic and Health Outcomes,” *Journal of Health Economics*, 33, pp. 76–93.

Lin, M.-J. and E. Liu (2014) “Does In Utero Exposure to Illness Matter? The 1918 Influenza Epidemic in Taiwan as a Natural Experiment,” *Journal of Health Economics*, 37, pp. 152–163.

Lleras-Muney, A. (2010) “The Needs of the Army Using Compulsory Relocation in the Military to Estimate the Effect of Air Pollutants on Children’s Health,” *Journal of Human Resources*, 45, No. 3, pp. 549–590.

Luechinger, S. (2014) “Air Pollution and Infant Mortality: A Natural Experiment from Powerplant Desulfurization,” *Journal of Health Economics*, 37, pp. 219–231.

Maccini, S. and D. Yang (2009) “Under the Weather: Health, Schooling, and Economic Consequences of Early-Life Rainfall,” *American Economic Review*, 99, No. 3, pp. 1006–1026.

Moretti, E. and M. Neidell (2011) “Pollution, Health, and Avoidance Behavior: Evidence from the Ports of Los Angeles,” *Journal of Human Resources*, 46, No. 1, pp. 154–175.

Nilsson, P. (2017) “Alcohol Availability, Prenatal Conditions, and Long-Term Economic Outcomes,” *Journal of Political Economy*, (forthcoming).

Persico, C., D. Figlio, and J. Roth (2016) “Inequality Before Birth: The Developmental Consequences of Environmental Toxicants,” *NBER Working Paper*, 22263.

Pope, C. A. (1986) “Respiratory Disease Associated with Community Air Pollution and a Steel Mill, Utah Valley,” *American Journal of Public Health*, 79, pp. 623–628.

Rundell, K. W. (2012) “Effect of Air Pollution on Athlete Health and Performance,” *British Journal of Sports Medicine*, 46, pp. 407–412.

Sanders, N. (2012) “What Doesn’t Kill You Makes You Weaker Prenatal Pollution Exposure and Educational Outcomes,” *Journal of Human Resources*, 47, No. 3, pp. 826–850.

Sanders, N. and C. Stoecker (2015) “Where Have All the Young Men Gone? Using Sex Ratios to Measure Fetal Death Rates,” *Journal of Health Economics*, 41, pp. 30–45.

Schlenker, W. and R. Walker (2016) “Airports, Air Pollution, and Contemporaneous Health,” *Review of Economic Studies*, 83, No. 2, pp. 768–809.

Sullivan, D. (2016) “The True Cost of Air Pollution: Evidence from House Prices and Migration,” *Working Paper (2016) available at http://scholar.harvard.edu/dsullivan/publications/cost-of-air-pollution.*

Tanaka, S. (2015) “Environmental Regulations on Air Pollution in China and Their Impact on Infant Mortality,” *Journal of Health Economics*, 42, pp. 90–103.

Wehby, G., D. Dave, and R. Kaestner (2016) “Effects of the Minimum Wage on Infant Health,” *NBER Working Paper*, 22373.

Wilkins, E. and Ministry of Health (1954) “Air Pollution Aspects of the London Fog of 1952,” in *Reports on Public Health and Medical Subjects*, London: Ministry of Health, pp. 267–271.

Yang, M. and S.-Y. Chou (2016) “Impacts of Being Downwind of a Coal-Fired Power Plant on Infant Health at Birth: Evidence from the Precident-Setting Portland Rule,” *NBER Working Paper*, 21723.

Table 1: Summary Statistics for Long-Term Outcomes

	All Cohorts		**1953 Cohort**	
	London	England & Wales	London	England & Wales
Fixed at conception				
Percentage male birth	0.49	0.50	0.50	0.50
Percentage non-white	0.04	0.01	0.03	0.01
Potential outcomes from exposure				
Holds degree in 1981	0.11	0.09	0.10	0.09
Unemployed in 1971	0.02	0.03	0.04	0.04
Unemployed in 1981	0.05	0.07	0.05	0.06
Unemployed in 1991	0.04	0.04	0.01	0.02
Unemployed in 2001	0.02	0.03	0.03	0.02
Unemployed in 2011	0.04	0.03	0.03	0.04
Hours worked in 1971	40.5	41.9	39.7	42.1
Hours worked in 1991	35.9	35.5	35.5	35.5
Hours worked in 2001	38.1	38.3	38.1	38.6
Permanently Sick in 1971	0.00	0.00	0.00	0.00
Permanently Sick in 1981	0.00	0.00	0.00	0.00
Permanently Sick in 1991	0.02	0.02	0.01	0.02
Permanently Sick in 2001	0.04	0.06	0.03	0.06
Permanently Sick in 2011	0.06	0.08	0.09	0.10
Died before 2011	0.03	0.03	0.02	0.04
Cancer registration before 2011	0.09	0.10	0.09	0.10
Observations	5,859	31,320	722	3,853

Notes : Data from the Office of National Statistics Longitudinal Study.

Table 2: Summary Statistics for Environmental and Pollution Data

	mean	std.dev	min	max
Quarterly minimum temperature	0.4	1.4	-3.6	4.3
Quarterly Maximum temperature	13.6	5.6	2.9	24.4
Quarterly Days of Frost	3.9	5.2	0.0	22.9
Quarterly Inches of Rain	54.5	29.0	2.3	145.0
Quarterly average black smoke	186.6	110.1	40.3	730.3

Notes: Temperatures measured in degrees Celsius. Weather data from the Met Office. Pollution data measured in microgrammes per meter cubed. Data from the Fuel Research Board.

Table 3: Summary Statistics for Short-Term Outcomes

	mean	std.dev	min	max
Births per 1000 people	4.6	4.4	0.2	34.3
Minimum temprature	0.4	1.4	-3.6	4.3
Quarterly average black smoke	185.9	53.7	100.6	350.7

Notes: Births data from the Registrar General for England and Wales. Temperatures measured in degrees Celsius. Weather data from the Met Office. Pollution data measured in microgrammes per meter cubed. Data from the Fuel Research Board.

Table 4: Regression Coefficients – Births Following the Smog

	(1) DD(1)	(2) DD(2)	(3) DD(3)	(4) DD(4)
Births following Smog	-0.09**	-0.09**	-0.09*	-0.09*
	(0.042)	(0.042)	(0.052)	(0.052)
Observations	789	789	789	789
Borough FE	no	yes	no	yes
Year FE	no	no	yes	yes

Notes: Dependent variable is births per one thousand people. Coefficients can be interpreted as the change in births per one thousand people. Robust standard errors are in parentheses, *** p<0.01, ** p<0.05, * p<0.1. Data on births from the Registrar General for England and Wales, data on weather from the MET office, data on black smoke pollution from the Fuel Research Board.

Table 5: Regression Coefficients – Infant Mortality Following the Smog

	(1) DD(1)	(2) DD(2)	(3) DD(3)	(4) DD(4)
Infant deaths in quarter of the Smog	0.01	0.01	0.01*	0.01*
	(0.006)	(0.006)	(0.007)	(0.006)
Infant deaths after the Smog	0.01	0.01	0.01**	0.01**
	(0.007)	(0.007)	(0.006)	(0.006)
Observations	789	789	789	789
Borough FE	no	yes	no	yes
Year FE	no	no	yes	yes

Notes: Dependent variable is deaths among infants less than one year old, per one thousand people. Coefficients can be interpreted as the change in infant deaths per one thousand people. Robust standard errors are in parentheses, *** p<0.01, ** p<0.05, * p<0.1. Data on births from the Registrar General for England and Wales, data on weather from the MET office, data on black smoke pollution from the Fuel Research Board.

Table 6: Regression Coefficients – Respondent is Male

	(1)	(2)	(3)	(4)
In Utero	-0.04		-0.03	
	(0.024)		(0.023)	
Infant	-0.06**		-0.05**	
	(0.024)		(0.023)	
Observations	459	459	459	459
Areas	51	51	51	51
Weather	yes	yes	yes	yes
Individual	no	yes	no	yes
Trends	no	no	yes	yes

Notes: Reported coefficients are estimates of the change in the percentage of males in the sample, caused by exposure to the Great London Smog while *in utero* or as an infant of less than one year old. Specifications (2) and (4) are omitted because the dependent variable contains individual information. Standard errors clustered at the area level are in parentheses, *** p<0.01, ** p<0.05, * p<0.1. All specifications include controls for levels and squares of black-smog particulate matter exposure and temperature. Fixed effects are included for year of birth and area of birth. Some specifications also include information on gender and ethnicity in each cell, and London, England and Wales time trends. Source: Office of National Statistics Longitudinal Study. Data on weather from the MET office, data on black smoke pollution from the Fuel Research Board.

Table 7: Regression Coefficients – Respondent is Not White

	(1)	(2)	(3)	(4)
In Utero	-0.01*		-0.00	
	(0.006)		(0.005)	
Infant	-0.03***		-0.02**	
	(0.007)		(0.006)	
Observations	459	459	459	459
Areas	51	51	51	51
Weather	yes	yes	yes	yes
Individual	no	yes	no	yes
Trends	no	no	yes	yes

Notes: Reported coefficients are estimates of the change in the percentage of people that are not white in the sample, caused by exposure to the Great London Smog *in utero* or as an infant of less than one year old. Specifications (2) and (4) are omitted because the dependent variable contains individual information. Standard errors clustered at the area level are in parentheses, *** p<0.01, ** p<0.05, * p<0.1. All specifications include controls for levels and squares of black-smog particulate matter exposure and temperature. Fixed effects are included for year of birth and area of birth. Some specifications also include information on gender and ethnicity in each cell, and London, England and Wales time trends. Source: Office of National Statistics Longitudinal Study. Data on weather from the MET office, data on black smoke pollution from the Fuel Research Board.

Table 8: Regression Coefficients – Degree-Level Qualification

	(1)	(2)	(3)	(4)
In Utero	-0.02	-0.02	-0.03**	-0.03**
	(0.012)	(0.012)	(0.014)	(0.013)
Infant	-0.00	-0.00	-0.02	-0.02
	(0.019)	(0.018)	(0.020)	(0.021)
Observations	459	459	459	459
Areas	51	51	51	51
Weather	yes	yes	yes	yes
Individual	no	yes	no	yes
Trends	no	no	yes	yes

Notes: Reported coefficients are estimates of the change in the percentage of people with a degree-level qualification, caused by exposure to the Great London Smog *in utero* or as an infant of less than one year old. Standard errors clustered at the area level are in parentheses, *** p<0.01, ** p<0.05, * p<0.1. All specifications include controls for levels and squares of black-smog particulate matter exposure and temperature. Fixed effects are included for year of birth and area of birth. Some specifications also include information on gender and ethnicity in each cell, and London, England and Wales time trends. Source: Office of National Statistics Longitudinal Study. Data on weather from the MET office, data on black smoke pollution from the Fuel Research Board.

Table 9: Regression Coefficients – Unemployment

	(1)	(2)	(3)	(4)
In Utero 1971	0.00	0.00	0.00	0.00
	(0.009)	(0.010)	(0.011)	(0.011)
Infant 1971	-0.01	-0.01	-0.01	-0.01
	(0.007)	(0.007)	(0.008)	(0.008)
In Utero 1981	0.01	0.01	0.00	0.01
	(0.019)	(0.019)	(0.020)	(0.020)
Infant 1981	0.01	0.01	-0.00	0.00
	(0.013)	(0.014)	(0.013)	(0.014)
In Utero 1991	-0.01	-0.01	-0.01	-0.01
	(0.010)	(0.009)	(0.009)	(0.009)
Infant 1991	-0.03***	-0.02**	-0.02***	-0.02**
	(0.009)	(0.009)	(0.007)	(0.007)
In Utero 2001	-0.00	0.00	0.00	0.00
	(0.006)	(0.006)	(0.006)	(0.006)
Infant 2001	-0.00	0.00	0.00	0.00
	(0.007)	(0.007)	(0.007)	(0.007)
In Utero 2011	-0.01	-0.01	-0.01	-0.01
	(0.007)	(0.007)	(0.008)	(0.007)
Infant 2011	-0.01	-0.01	-0.02	-0.02
	(0.008)	(0.009)	(0.012)	(0.012)
Observations	459	459	459	459
Areas	51	51	51	51
Weather	yes	yes	yes	yes
Individual	no	yes	no	yes
Trends	no	no	yes	yes

Notes: Reported coefficients are estimates of the change in the percentage of people who are unemployed, caused by exposure to the Great London Smog *in utero* or as an infant of less than one year old. Standard errors clustered at the area level are in parentheses, *** p<0.01, ** p<0.05, * p<0.1. All specifications include controls for levels and squares of black-smog particulate matter exposure and temperature. Fixed effects are included for year of birth and area of birth. Some specifications also include information on gender and ethnicity in each cell, and London, England and Wales time trends. Source: Office of National Statistics Longitudinal Study. Data on weather from the MET office, data on black smoke pollution from the Fuel Research Board.

Table 10: Regression Coefficients – Hours Worked

	(1)	(2)	(3)	(4)
In Utero 1971	-8.17**	-7.85**	-8.03**	-7.78**
	(3.442)	(3.422)	(3.302)	(3.338)
Infant 1971	-4.95**	-4.19*	-4.33**	-3.78*
	(2.314)	(2.313)	(1.912)	(1.995)
In Utero 1991	-1.79***	-1.27**	-1.68**	-1.24*
	(0.666)	(0.601)	(0.662)	(0.652)
Infant 1991	-1.03	-0.09	-0.85	-0.05
	(0.650)	(0.658)	(0.786)	(0.773)
In Utero 2001	-1.31	-0.82	-1.17	-0.76
	(0.842)	(0.724)	(0.810)	(0.714)
Infant 2001	-0.22	0.65	0.01	0.76
	(0.958)	(0.942)	(0.891)	(0.837)
Observations	459	459	459	459
Areas	51	51	51	51
Weather	yes	yes	yes	yes
Individual	no	yes	no	yes
Trends	no	no	yes	yes

Notes: Reported coefficients are estimates of the change in hours worked, caused by exposure to the Great London Smog *in utero* or as an infant of less than one year old. Standard errors clustered at the area level are in parentheses, *** p<0.01, ** p<0.05, * p<0.1. All specifications include controls for levels and squares of black-smog particulate matter exposure and temperature. Fixed effects are included for year of birth and area of birth. Some specifications also include information on gender and ethnicity in each cell, and London, England and Wales time trends. Source: Office of National Statistics Longitudinal Study. Data on weather from the MET office, data on black smoke pollution from the Fuel Research Board.

Table 11: Regression Coefficients – Permanently out of Work due to Health

	(1)	(2)	(3)	(4)
In Utero 1971	-0.00***	-0.00***	-0.00*	-0.00*
	(0.001)	(0.001)	(0.001)	(0.001)
Infant 1971	-0.00	-0.00	-0.00	-0.00
	(0.002)	(0.002)	(0.002)	(0.002)
In Utero 1981	-0.00**	-0.01**	-0.00**	-0.00**
	(0.002)	(0.002)	(0.002)	(0.002)
Infant 1981	0.00	-0.00	0.00	-0.00
	(0.002)	(0.003)	(0.003)	(0.003)
In Utero 1991	-0.00	-0.00	-0.00	0.00
	(0.006)	(0.006)	(0.005)	(0.005)
Infant 1991	0.00	0.00	0.00	0.00
	(0.005)	(0.006)	(0.006)	(0.006)
In Utero 2001	-0.02*	-0.02	-0.02*	-0.02*
	(0.009)	(0.009)	(0.010)	(0.010)
Infant 2001	0.00	0.01	0.00	0.00
	(0.009)	(0.010)	(0.010)	(0.011)
In Utero 2011	-0.01	-0.01	-0.01	-0.01
	(0.013)	(0.013)	(0.013)	(0.013)
Infant 2011	-0.01	-0.01	-0.02	-0.01
	(0.010)	(0.011)	(0.012)	(0.012)
Observations	459	459	459	459
Areas	51	51	51	51
Weather	yes	yes	yes	yes
Individual	no	yes	no	yes
Trends	no	no	yes	yes

Notes: Reported coefficients are estimates of the change in the probability of being permanently out of work due to illness, caused by exposure to the Great London Smog *in utero* or as an infant of less than one year old. Standard errors clustered at the area level are in parentheses, *** p<0.01, ** p<0.05, * p<0.1. All specifications include controls for levels and squares of black-smog particulate matter exposure and temperature. Fixed effects are included for year of birth and area of birth. Some specifications also include information on gender and ethnicity in each cell, and London, England and Wales time trends. Source: Office of National Statistics Longitudinal Study. Data on weather from the MET office, data on black smoke pollution from the Fuel Research Board.

Table 12: Regression Coefficients – Cancer Diagnosis

	(1)	(2)	(3)	(4)
In Utero	0.00	-0.00	0.00	0.00
	(0.014)	(0.014)	(0.014)	(0.014)
Infant	0.03	0.02	0.03*	0.03*
	(0.017)	(0.017)	(0.016)	(0.016)
Observations	459	459	459	459
Areas	51	51	51	51
Weather	yes	yes	yes	yes
Individual	no	yes	no	yes
Trends	no	no	yes	yes

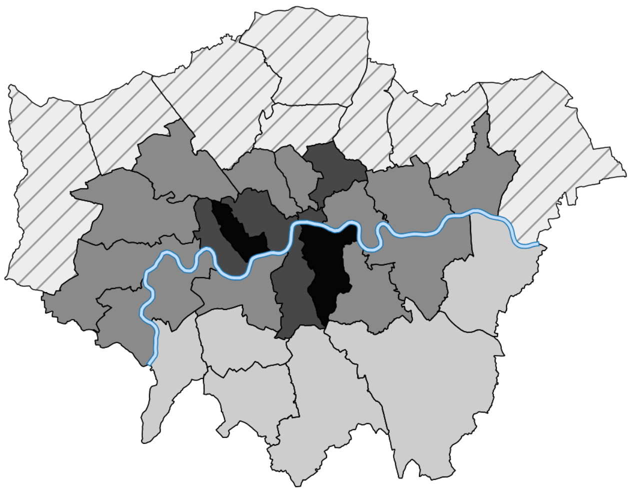
Notes: Reported coefficients are estimates of the change in the probability of developing a cancer, caused by exposure to the Great London Smog *in utero* or as an infant of less than one year old. Standard errors clustered at the area level are in parentheses, *** p<0.01, ** p<0.05, * p<0.1. All specifications include controls for levels and squares of black-smog particulate matter exposure and temperature. Fixed effects are included for year of birth and area of birth. Some specifications also include information on gender and ethnicity in each cell, and London, England and Wales time trends. Source: Office of National Statistics Longitudinal Study. Data on weather from the MET office, data on black smoke pollution from the Fuel Research Board.

Table 13: Regression Coefficients – Died before Age Sixty

	(1)	(2)	(3)	(4)
In Utero	-0.02***	-0.02***	-0.02***	-0.02***
	(0.006)	(0.006)	(0.006)	(0.006)
Infant	-0.02**	-0.02**	-0.02*	-0.02*
	(0.009)	(0.010)	(0.010)	(0.011)
Observations	459	459	459	459
Areas	51	51	51	51
Weather	yes	yes	yes	yes
Individual	no	yes	no	yes
Trends	no	no	yes	yes

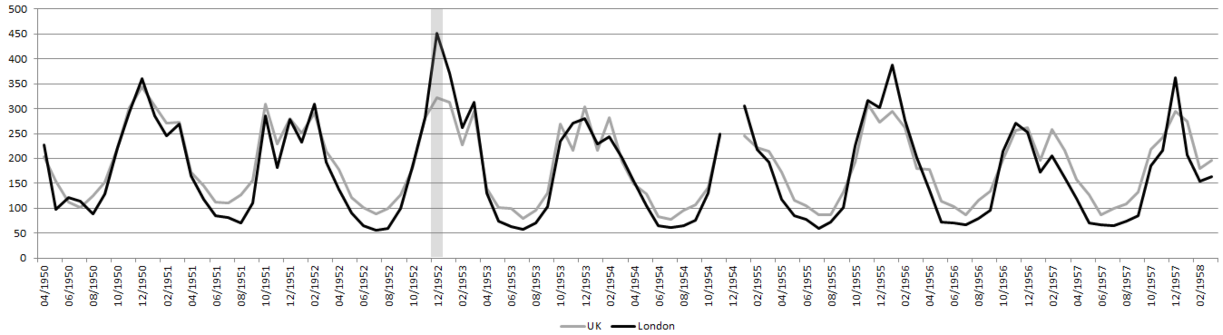
Notes: Reported coefficients are estimates of the change in the probability of dying before 2011 (or the age of around sixty, for those affected by the smog), caused by exposure to the Great London Smog *in utero* or as an infant of less than one year old. Standard errors clustered at the area level are in parentheses, *** p<0.01, ** p<0.05, * p<0.1. All specifications include controls for levels and squares of black-smog particulate matter exposure and temperature. Fixed effects are included for year of birth and area of birth. Some specifications also include information on gender and ethnicity in each cell, and London, England and Wales time trends. Source: Office of National Statistics Longitudinal Study. Data on weather from the MET office, data on black smoke pollution from the Fuel Research Board.

Figure 1: Impact of the Smog in December 1952



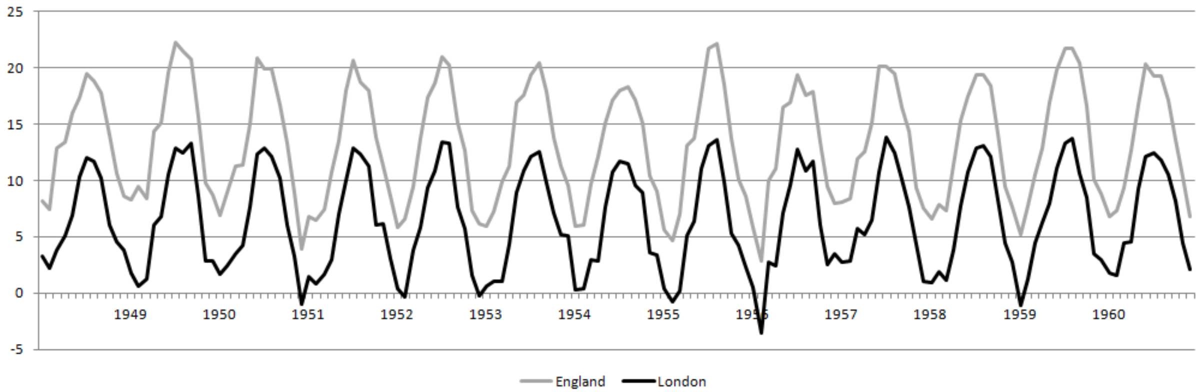
Notes: Figure shows Greater London divided into Boroughs, with the Thames running from west to east. For reference, London Boroughs average around 40 square kilometres in size, and had average populations of around 100,000 at the time of the smog. The shading is based on a figure published in Wilkins (1954) in which information from 117 sulphur dioxide instruments was used to divide London into four approximate levels of impact, with a legend reported in arbitrary units. The shading in the figure is therefore relative, with the lightest grey serving as a baseline, medium grey representing levels twice as high, dark grey representing levels three times as high, and black indicating levels that are more than three times the baseline. The two darkest regions include the London Boroughs of Kensington and Chelsea, Southwark, Lambeth, Westminster, Hackney, Hammersmith and Fullham, and the City of London. The two lighter regions include Newham, Wansworth, Barking and Dagenham, Brent, Camden, Ealing, Greenwich, Hounslow, Islington, Lewisham, Richmond, Tower Hamlets, Bexley, Bromley, Croydon, Kingston, Merton, and Sutton. There is no information on exposure in the striped area to the north. (Though the area was affected by the Smog.) Pollution data from the Fuel Research Board and Wilkins (1954), mapping data from the Ordinance Survey.

Figure 2: Monthly Average Levels of Particulate Pollution (Black Smoke) in London and the U.K.



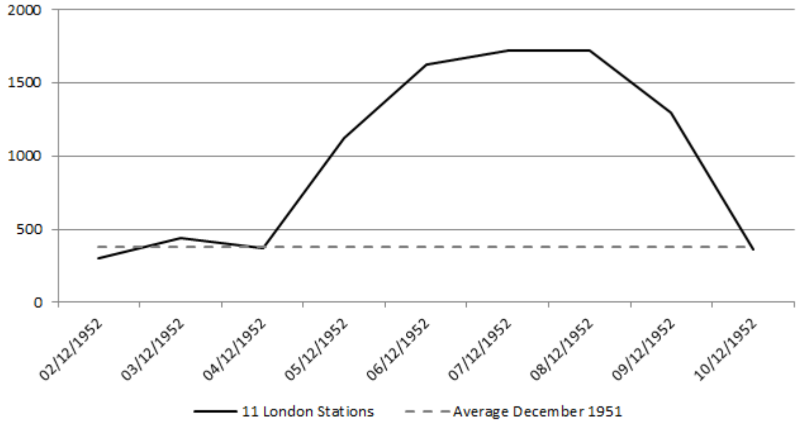
Notes: Particulate pollution measured in microgrammes per meter cubed, black smoke. London series based on fifteen daily meters in the Greater London area. UK series based on fifty eight urban daily meters. Data from the Fuel Research Board.

Figure 3: Daily minimum temperatures in London and England, degrees Celsius



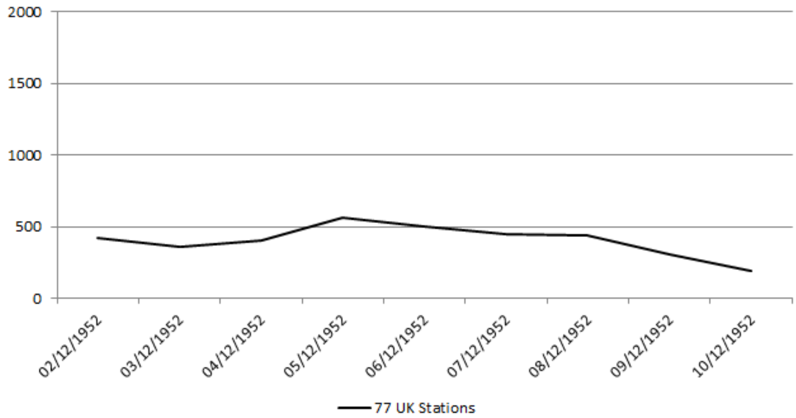
Notes: Temperatures measured in degrees Celsius. Data from the Met Office.

Figure 4: Daily Average Pollution in December 1952 - London



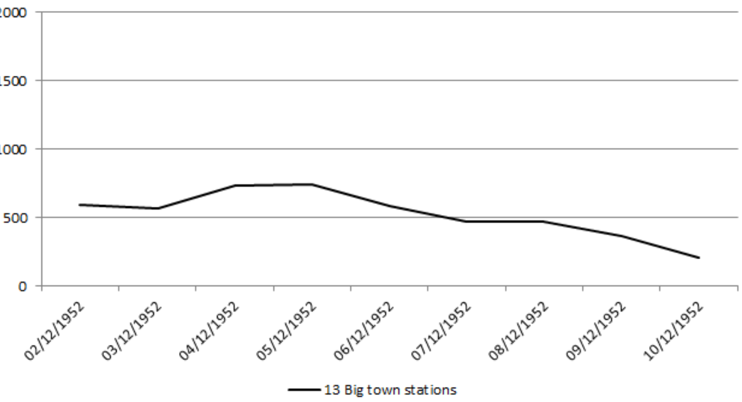
Notes: Pollution data is for black smoke particulates and is measured in microgrammes per cubic metre. Solid black line based on an average from 11 daily monitoring stations in London. Data from the Fuel Research Board.

Figure 5: Daily Average Pollution in December 1952 - Great Britain (Excluding London)



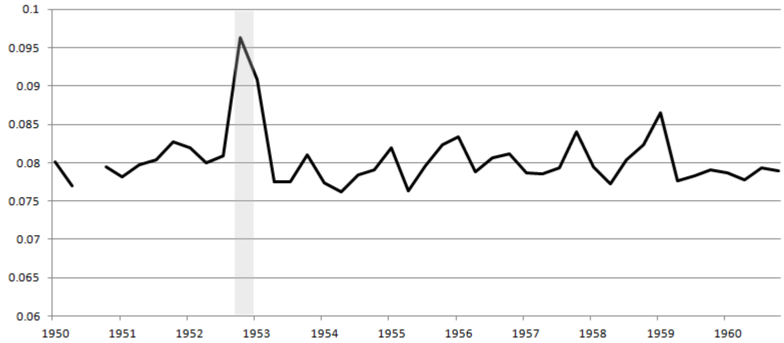
Notes: Pollution data is for black smoke particulates and is measured in microgrammes per cubic metre. Solid black line based on an average from 77 daily monitoring stations in Great Britain. Data from the Fuel Research Board.

Figure 6: Daily Average Pollution in December 1952 - Other Big Towns (Excluding London)



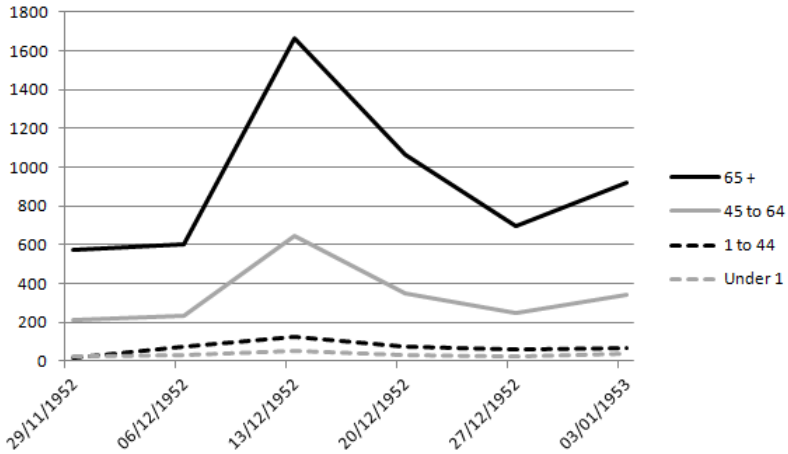
Notes: Pollution data is for black smoke particulates and is measured in microgrammes per cubic metre. Solid black line based on an average from 16 daily monitoring stations other large towns in the UK. These are: Bradford, Bristol, Cardiff, Glasgow, Leicester, Liverpool, Manchester, Newcastle, Nottingham, and Sheffield. Data from the Fuel Research Board.

Figure 7: Ratio of Deaths in London to Deaths in England and Wales



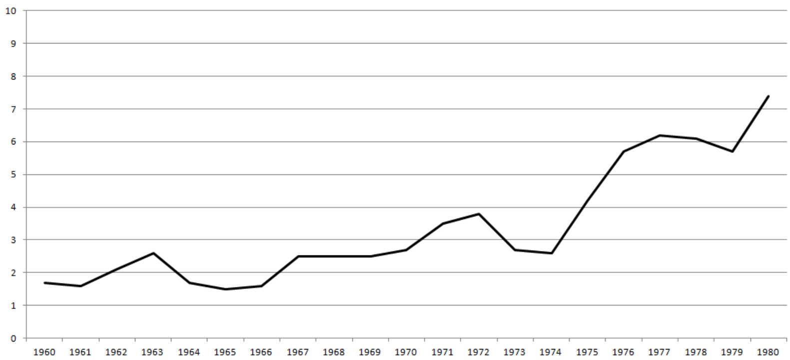
Notes: Ratio of quarterly deaths in London to quarterly deaths in England and Wales. Data from the Registrar General for England and Wales

Figure 8: Deaths in London during the Smog of 1952, by Age



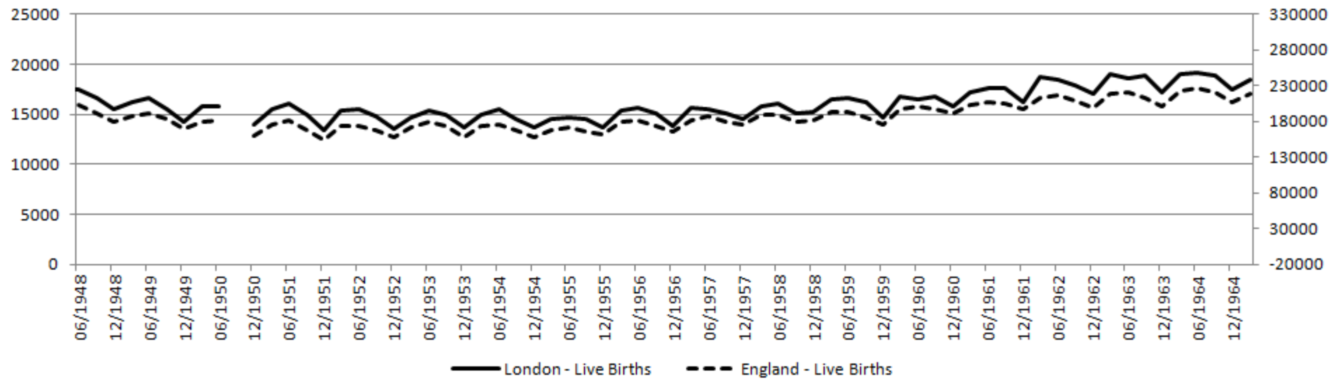
Notes: Deaths reported at the end of each week to the London administrative county, recorded in Ministry of Health (1954)

Figure 9: Labour Market Conditions (Unemployment) in the UK



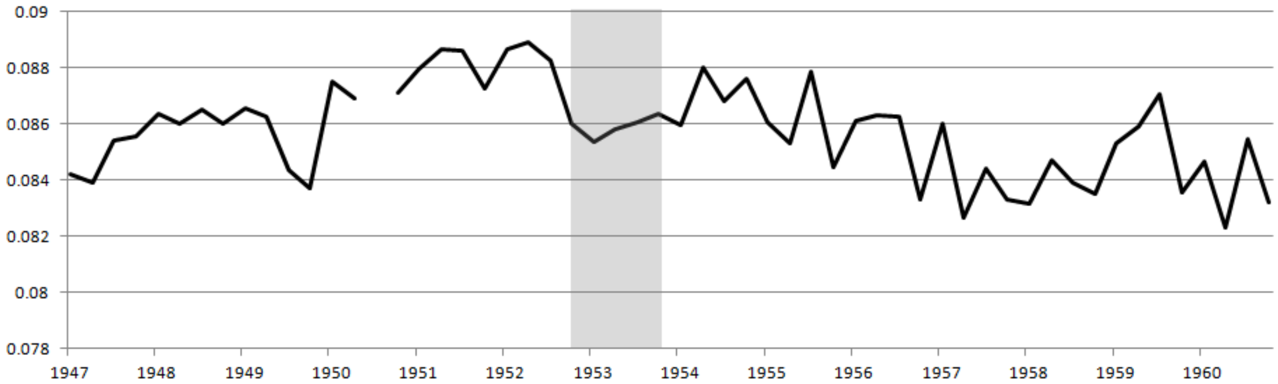
Notes: The (affected) 1952 cohort would enter the labour market between 1969 and 1971 when average levels of unemployment were 2.8% on average. The (unaffected) 1953 cohort would enter the labour market between 1970 and 1972 when average levels of unemployment were 3.1% on average. Source: Office of National Statistics

Figure 10: Births in London (left axis), England & Wales (right axis)



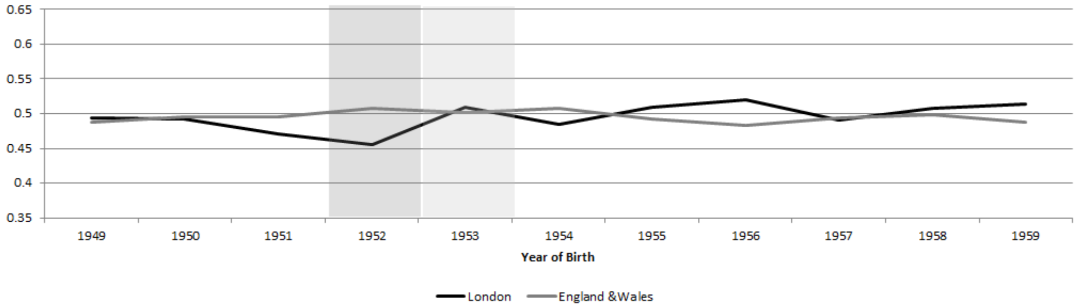
Notes: Data from the Registrar General

Figure 11: Ratio of Births in London, and England & Wales



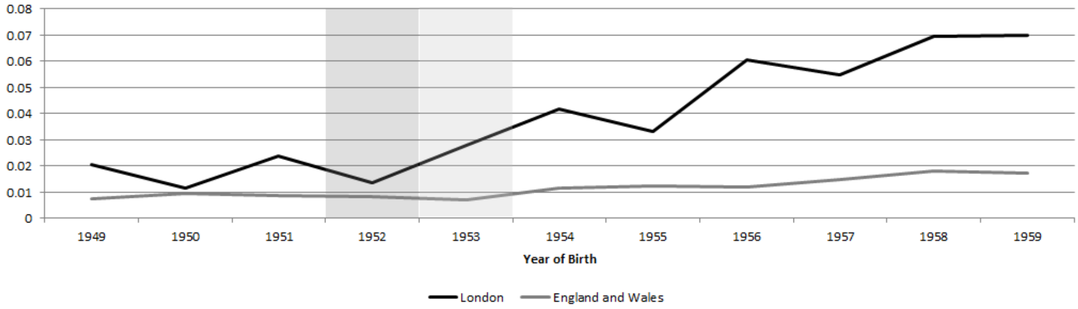
Notes: Data from the Registrar General

Figure 12: Respondent is Male



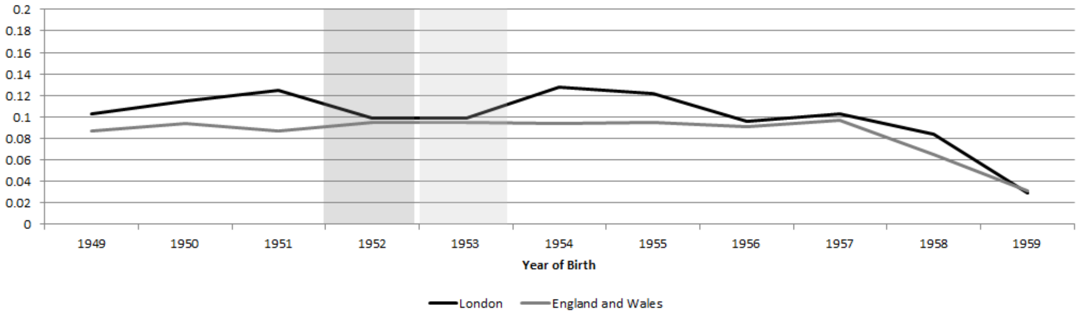
Notes: Those who were infants in London during the Great London Smog are highlighted in Dark Gray. Those who were *in utero* highlighted in light gray. Data from the Office of National Statistics Longitudinal Study

Figure 13: Respondent is Non-White



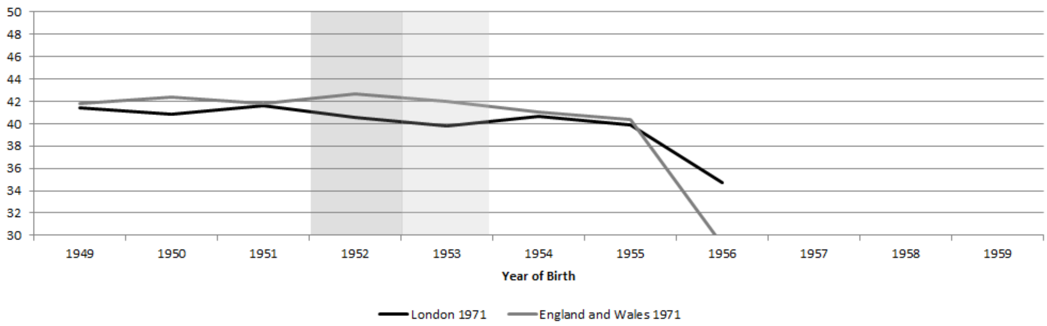
Notes: Those who were infants in London during the Great London Smog are highlighted in Dark Gray. Those who were *in utero* highlighted in light gray. Data from the Office of National Statistics Longitudinal Study

Figure 14: Respondent Has a Degree



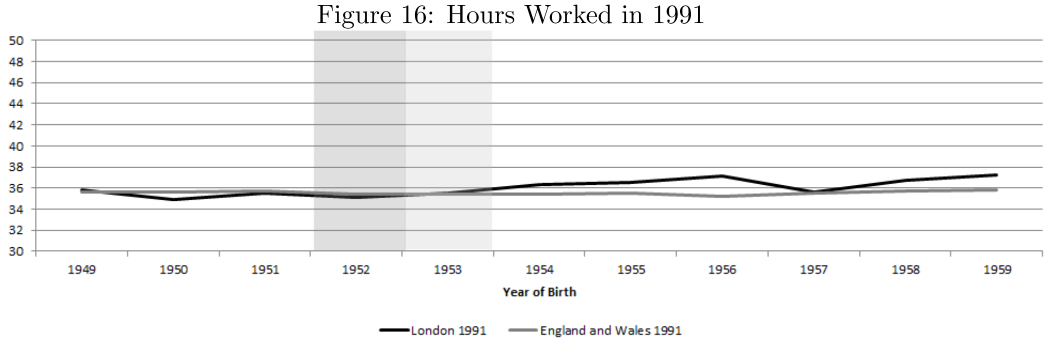
Notes: Whether someone has a degree is determined by whether they report as having a degree in any of 1971, 1981, 1991, or 2001. Those who were infants in London during the Great London Smog are highlighted in Dark Gray. Those who were *in utero* highlighted in light gray. Data from the Office of National Statistics Longitudinal Study.

Figure 15: Hours Worked in 1971



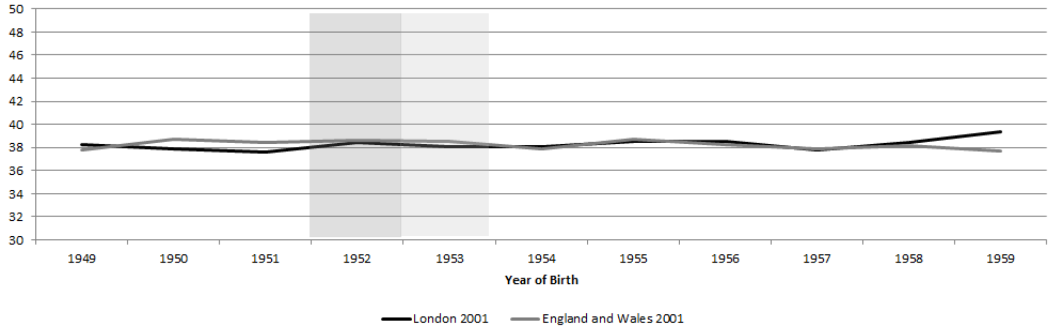
Notes: Those who were infants in London during the Great London Smog are highlighted in Dark Gray. Those who were *in utero* highlighted in light gray. Data from the Office of National Statistics Longitudinal Study.

Figure 16: Hours Worked in 1991



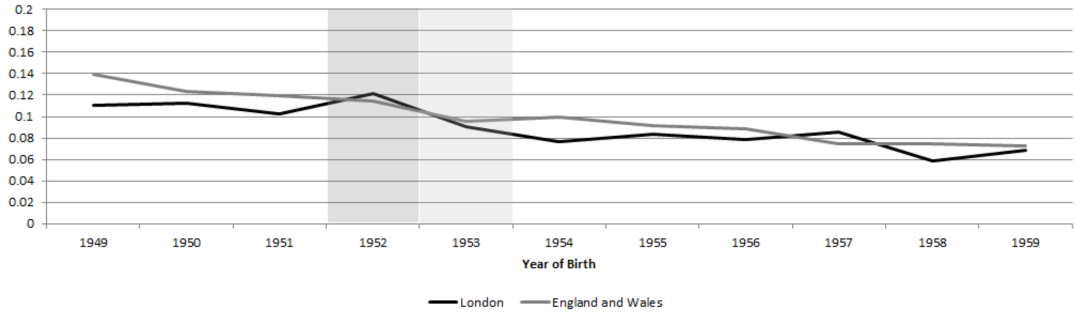
Notes: Those who were infants in London during the Great London Smog are highlighted in Dark Gray. Those who were *in utero* highlighted in light gray. Data from the Office of National Statistics Longitudinal Study.

Figure 17: Hours Worked in 2001



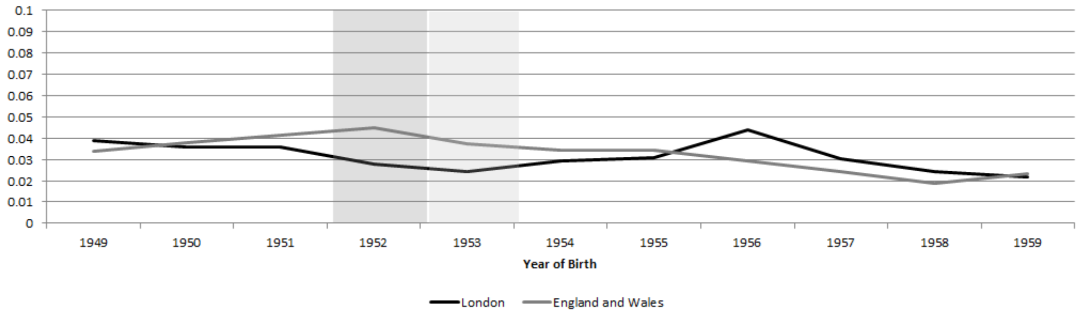
Notes: Those who were infants in London during the Great London Smog are highlighted in Dark Gray. Those who were *in utero* highlighted in light gray. Data from the Office of National Statistics Longitudinal Study

Figure 18: Respondent Has Registered Cancer



Notes: Those who were infants in London during the Great London Smog are highlighted in Dark Gray. Those who were *in utero* highlighted in light gray. Data from the Office of National Statistics Longitudinal Study.

Figure 19: Respondent Died Before 2011



Notes: Those who were infants in London during the Great London Smog are highlighted in Dark Gray. Those who were *in utero* highlighted in light gray. These cohorts would have been around sixty years old in 2011. Data from the Office of National Statistics Longitudinal Study.